٢١st/Feb/٢٠١١

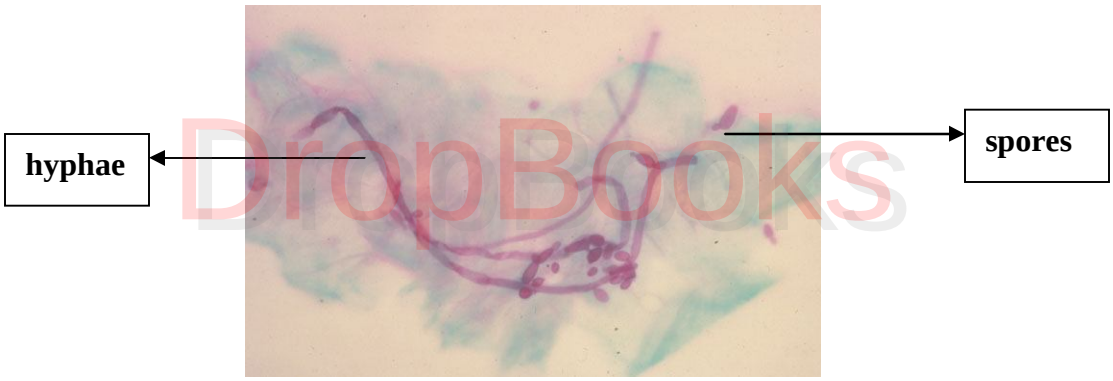
Oral patho #٢
Fungal infection and HIV

Appreviations :
Oral cavity : o.c,, candida albicans : cand.A ,,epithelium:epith

✳Candida's structure and characteristics✳

✶The most common organism causing fungal infections in the o.c is **CANDIDA** and the most common species of candida is called **ALBICANS**

✶If u look at the picture of cand.A, u'll see that this organism is **dimorphic** : meaning that it exists in ٢ forms :
١-the hyphae: which's a long thread like .
٢-the spores : which is rounded and those are responsible for multiplying by budding.



✶Cand.A is considered **commensal**: meaning that it lives in the oc peacefully without causing diseases, but it's also **opportunistic** "entehazyeh" meaning that if there is a good situation for this organism to induce infection, it will not stop ,it will cause infection and the situations that allow it to cause infection are those that cause disturbance in the homeostatic balance.

….. *note: homeo and not hemo; homeo :meaning home conditions
hemo : related to blood
So, it's important to have a homeostatic balance between the host and the organisms and a homeostatic disturbance could cause it to start acting as a pathogen.

✶ we have an interplay between the pathogens of the oc "bacteria and fungus" and our defensive mechanisms ,, these mechanisms may be nonspecific meaning acting in the same way against anything present in the oc; bacteria, fungus or viruses :

☞Nonspecific defensive mechanisms in the oc :

١-saliva: salivary flow has a washing effect for on candida, bacteria ,foreign bodies or what ever.

٢- sloughing or desquamation of oral epith. : there is a cycle present normally for the regeneration of the oral epith. Which leads to loss of organisms that may organize on it.

٣-the phagocytic capacity of the neutrophiles and the macrophages.

٤-the presence of bacteria in the oc is also important as a defensive mechanism against cand.,, we should have a balance between the bacterial count and the candidal count, so, if a person takes a broad spectrum of antibiotics for say ٢ weeks ,the bacterial count will go down ,as a result, the fungal count will increase and the shape of cand. Will Change to cause infection.

☞Specific defensive mechanisms in the oc sagainst cand.:

١-serum antibodies :defense of these antibodies is low against cand.

٢-the secretory antibodies **like IgA** secreted in saliva : secretory antibodies Are more important than serum anibodies.

٣-cell mediated immunity : which is more specific for cand. And this is proven by the fact that the chronic candidal Infection is found in people with impaired mediated cell immunity ,so, cell mediated immunity is important to prevent candidal infection and that's why the people with chronic candidoses happened to have impaired immunity specifically the cell mediated one. People with impaired cell mediated immunity could get also systemic fungal infections other than oral infections such as :
chronic cutaneous fungal infections or mucocutaneous cadidoses

☞The factors that cause the cand. To change into a pathogen :
We said that the secretory IgA and the cell mediated immunity are important to fight against candidal Infections and so, the factors that lead to immune system suppression will also lead to fungal infection , give me factors that may lead to immune system suppression : students' answers :
١-stress ٢-transplntation ٣-cytotoxic medications ٤-autoimmune diseases ٥-AIDS and HIV ٦-steroids ٧-pregnancy ٨-diapetes

☞So, We have factors that cause disturbance in homeostatic balace:

✶Local factors:

- 🖋Trauma
- 🖋Denture hygiene: if the pnt didn't clean the fitting surface of the denture there will be a good area for colonization cuz this will prevent the washing effect of saliva from reaching the palate or the denture surface.
- 🖋Ill-fitting or rocking denture also will induce the continuous trauma of the palate and surface epith.
- 🖋Tobacco smoking : this will change the whole environment and temperature and it may predispose to other diseases other than candidoses.
- 🖋Carbohydrate- rich diet : people who eat a lot of sugar could become infected with cand

✶age : the ٢ extremes :very young "infants" or elderly

✶drugs:

١-broad spectrum of antibiotic

٢-steroids

٣-cytotoxic medication

✶Xerostomia : the washing effect of saliva will be lost so, the cand will have the opportunity to colonize on the epith.surface.

✶Other systemic diseases

✶carriage rate of cand in the oc is variable among people, more than ٤٠٪ of people will carry cand. In their mouths , but the count of cand is **NOT** an indicator of the disease but **the shape or form of the cand** is . cuz if we examine a pnt with candidoses and count the cand, the count won't be different from that who doesn't have cadidoses, so, sometimes the carrier person and the infected have the same count of cand.

✶the hyphal form in the smear is important for our diagnosis ; it was found that the **hyphal form** of cand has more role in causing the infection than the ovoid from. cuz it's the one that would invade and adhere the **surface layer** of the oral epith. And the ovoid is important in budding and replication, so, in order for us to diagnose a pnt with candidoses ,the hyphal form of cand should be found in his mouth.

✶the storage area of cand is **the dorsum of the tongue** .

✳Pathogenesis✳

✶What's the mechanism by which cand switch to being a pathogen ??
☑ First it should have a genetic switch ; spores switch from rounded organisms to hyphae , and this happens due to the factors we talked about.

☑Then hyphae adheres to the oral mucosa.

☑Then it secretes enzymes that start loosening the intercellular bridges between the epith. cells to allow the hyphae to be imbedded in between, however , the hyphae will NOT insert itself for long distances within the epith.
It will only stay on the superficial epith.layer which is the parakeratin and part of the prickle cell layer and will not reach the basal layer,,
but to induce the inflammatory response ,its effect should go all the way down the epith. to reach the submucosa , this is accomplished by the inflammatory materials produced by the hyphae, these materials or enzymes will reach the submucosa and the immune system will be activated,

Note >> Cand can secrete : ***nitrosamine*** which is a carcinogenic agent that can be found in tobacco, So , cand may produce premalignant lesions cuz it produces nitrosamine , ,
if u remember we have a variant of leukoplakia called candidal leukoplakia which is at a higher risk of developing carcinoma compared to homogenous leukoplakia, cuz most of candidal leukoplakia are nonohomogenous; it can be nodular , erythroplakia or spekled, so once we see a candidal leukoplakia we should be worried about developing a cancer; cuz of the presense of nitrosamine produced by cand.
Some theories are against this and suggest that the candidal Leukoplakia was homogenous leukoplakia but got secondarly infected by cand; cuz cand. In general Likes rough surfaces and the dorsum of the tongue, so, they assume that the cand selected these places to colonize it, so ,it is a result and not a cause, ,
but then again ,others say that if u apply an antifungal agent on these lesions some may regress, so, cand might have something to do with the etiology of cand leukoplakia .
regardless of all when ever u hear about cand. Leukoplakia u should know it's most likely: **nonhompgenous** and **at higher risk of developing cancer** compared to homogenous one cuz of the secretion of nitrosamine by the fungal hyphae.

Note>> The cand products may produce a hypersensitivity reaction in our body and the immune system products will start coming up and reaching to below the epith. Surface or even within the surface .
Suppose that u suspect that the pnt has candidoses on the dorsum of the tongue ,so u do a Smear preperation by scrapping the lesion with a wooden spatula and spread it on the smear ,u may fix it by alcohol ;u can use the hair spray cuz it's rich with alcohol and then send it to the lab and ask for a PAS stain, which will show u the fungl hyphae, if the u didn't see hyphae but spores only, then u'll say that the pnt is only a carrier and not having a fungal infection .

Immune system products going up to the epith.and start a hypersensitivity reaction

Hyphae Can only reach to part of the prickle cell layer and not further below

Cand may produce premalignant lesions due to nitrosamine
Cand may produce hypersensitivity reaction due to immune system

✳classifications of candidoses✳

We have :

- ✶Acute: can be white or red.
- ✶Chronic: can be white or red
- ✶candida associated lesions
- ✶ secondary oral candidoses.

- Acute
  - White "psuedomembranous"= thrush
  - Red "erythematous or atrophic*"= antibiotic sore tongue
- chronic
  - White "psuedomembranous"= in AIDS pnts
  - Red "erythematous or atrophic*" = cand. Associated denture stomatitis
  - White "hyperplastic (candidal leukoplakia)"

atrophic : when the epith intraorally is **atrophic its color becomes **red** ,but when it's **hyperplastic**, its color becomes **white**, cuz the shadow of the blood vessels of the submucosa will appear better if the epith is atrophic and it will appear less if the epith is hyperkertinized or hyperplastic,, so, if the epith is atrophic then the lesion is erythematous but if it's thick, it will be white and called psuedomembranous.

◈acute pseudomembranous candidoses :

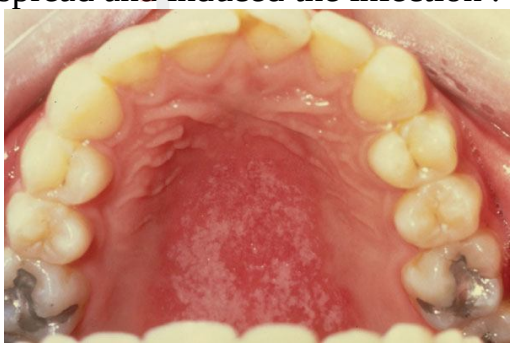
✶Also called **THRUSH**

✶It's common in neonates, elderly ,pnts after medication or antibiotics, pnts with reduced immunity and all the factors we already mentioned that may disturb the homeostasis in the oc.

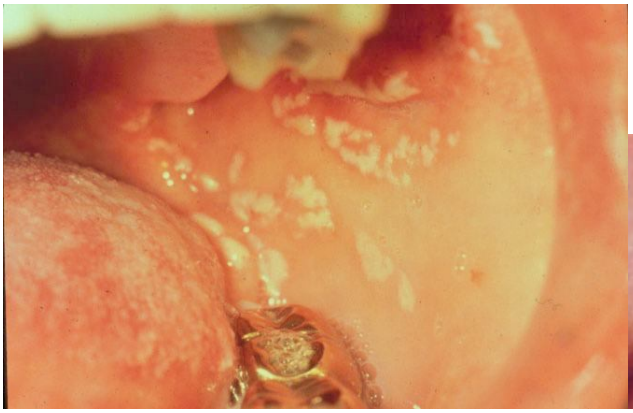
✶it can be removed easily by scrapping , but below it we'll have painful areas and red bleeding bases .

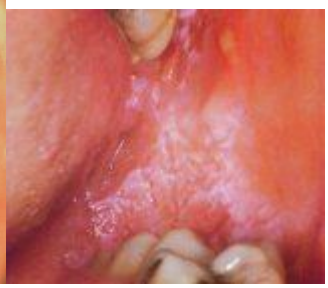
✶it's widely spread.

✶it may be found on any mucosal surface in the mouth; the buccal mucosa or the palate…, lets say u saw them on the palate ,u may say these are only desquamating epith cells and u may remove them and they will go away , but we must ask the pnt what made them desquamating ?
I will ask about mouth wash , if we find this lesion say on the lower lip, we ask the pnt if he continuously bites it and …ets
if none of the above, we take a smear, put it on the slide ,send it to the lab and u will see the hyphae, especially when this pnt had this lesion and another one on the tongue and by contact btw the palate and the tongue, the organisms may have spread and induced the infection .



✂Psuedomembranous lesion Vs aspirin burn.
Look at the picture below, at the buccal mucosa ,, u may think of **aspirin burn** ,but if the pnt used aspirin as a mouth wash and spread it all over ,other areas of the oc besides the buccal mucosa should be involved ,and we can't say that the pnt has applied topical aspirin cuz the lesion is wider than that.





Psuedomembranous lesion aspirin burn

✂Psuedomembranous lesion Vs leukoplakia.

Pseudomembranous has a necrotic material which's white , leukoplakia by definition is a white lesion that can't be removed by scrapping, while pseudomembranous is a white lesion that can be removed by scrapping , so if we see a white lesion that can be removed, we think of candidoses ,aspirin burn , chemical burn, thermal burn ,trauma ,cheek biting …ets. U have to think about a variety of causes for this psuedomembrane ,but if it can't be removed by scrapping and doesn't diappear upon stretching , it can be any kind of leukoplakia , it can be hyperplastic candidoses which's in other words candidal leukoplakia , so u have to think about another way of differential diagnosis.

◈chronic pseudomembranous candidoses :

The chronic pseudomembranous is the same as the acute, but:

- it's seen more in the immunocompromised pnts especially the AIDS pnts
- it involves more than one area generally affecting the whole oc
- doesn't respond to antifungals; for acute, the lesion disappears after application of antifungals in ١٠ days or so, but in the chronic one in AIDS ,the pnt doesn't respond to antifungals.

◈acute erythematous candidoses :

✶also called acute atrophic candidoses or antibiotic sore tongue

✶Here we have a relatively atrophic area on the **dorsum of the tongue**, the papillary are lost, it's slightly red so this is called erythematous candidoses .

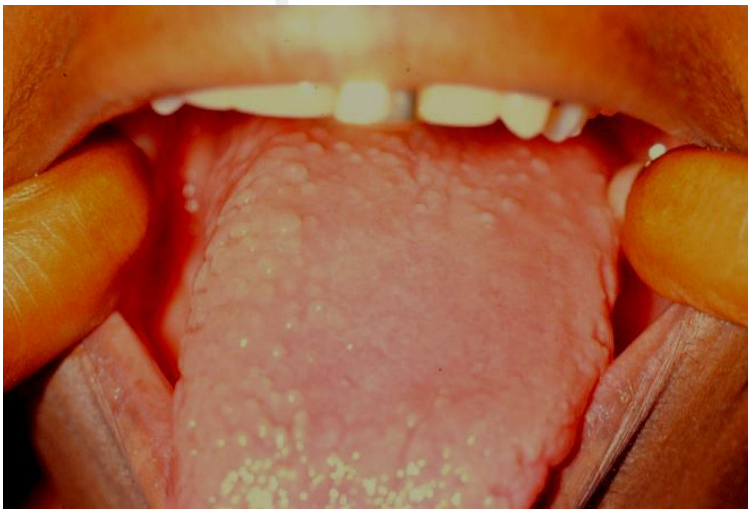
✶ it's acute , why is it acute ? cuz within ٢ weeks the pnt may have taken a broad spectrum of antibiotics and after finishing the course he will start having this painful atrophic area on the dorsum of the tongue and it's called acute erythematous candidsoses .

✶the pnt will have burning sensation and erythema.

✶ and it USUALLY happens after antibiotics, and that's why it's called antibiotic sore tongue, but other causes may induce this type ,but most commonly it's associated with broad spectrum of antibiotics.

✶ it's red and relatively more painful than the thrush .

so ,it's very important to take history of the pnt and ask about the intake of antibiotics.



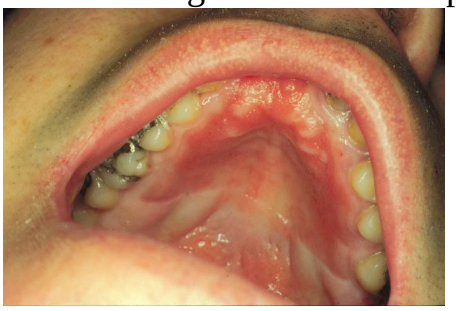
◈chronic erythematous candidoses :

✶also called: candida associated denture stomatitis or chronic atrophic candidoses.

✶so, acute atrophic candidoses>> is antibiotic sore tongue ,
the chronic atrophic >> is candida associated denture stomatitis

✶a chronic use of something may Induce candidoses, and here ,is a chronic use of an upper denture covering a wide area of the oral mucosa, when there is a continuous wear of the upper denture and the oral hygiene is poor or the denture may be rocking ,these all give the candida time to colonize.

✶ the interesting thing is that the cand doesn't colonize the mucosal surface, it only colonizes the **FITTING SURFACE** of the denture ,so, what's the mechanism of inducing this red atrophic lesion on the palate?? it may be a hypersensitivity reaction on the mucosal surface in response to cand found on the fitting surface of the denture ,so the cand in the chronic trophic candisdoses doesn't colonize on the dorsum of the tongue or the palate or what ever it only colonizes the fitting surface of the upper denture.



✶ which is more common to have a chronic atrophic candidoses, the upper or the lower? The **UPPER** ,,cuz it's covering a wider area.

✶ we have ٣ patterns of candida associated denture stomatitis depending on ***newton's classification***:

☛ the mildest one is pinpointed erythema; grade ١ .

☛diffused erythema ;grade ٢. "the one in the picture above."

☛ the granular or multinodular ;grade ٣, here we'll have ***chronic inflammatory papillary hyperplasia,*** if u remember we took it in connective tissue !! "yeah right!!" we said it's associated with candida and it's the ٣rd grade of newton's classification and it's the worst cuz it needs surgical removal of the papillae cuz the granules that form will interfere with the fitting of the denture.

So, here we have the same principle as leukoplakia, the more nodular the lesion, the more severe,

◈chronic hyperplastic candidoses :

✶ it's also called candidal leukoplakia.

✶it's white and unlike thrush it can't be removed by scrapping cuz it's hyperplastic.

✶The commeasures and the buccal mucosa are the most commonly affected sites.

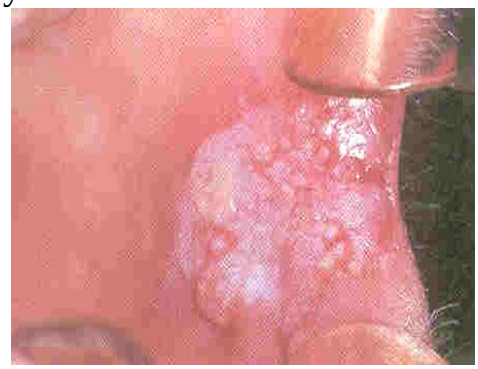
✶ looks roughly triangular with its base on the corner and its tip pointing posteriorly.

✶this is usually found in heavy smokers , and is common in our clinic ; we give the pnt an antifungal and ask him to come back in ١٤ days.

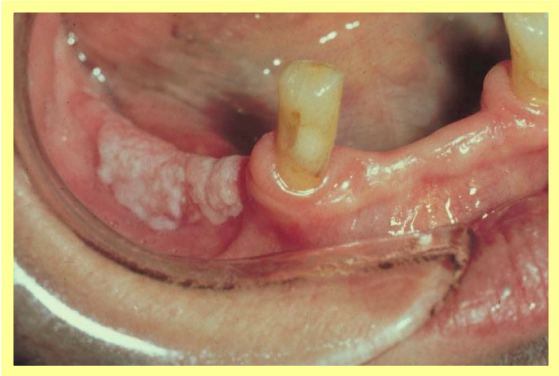
✶It's red and white, nonhomogenous ,erythroplakia may be seen, nodular or spekled and if u take a biopsy u'll see hyphae invading the superficial layer of the surface epith.

✶And cuz it happens at the commissures it's usually associated with **angular chilitis;** which's an inflammation at the cornenrs of the mouth
✶It Can be multifocal and called ***chronic multifocal oral candidoses*** and pnts with this disease have impaired cell mediated immunity ,so these pnts get both thrush and hyperplastic candidoses .

✶ hyperplastic candidoses is the most common form in pnts with impaired cell mediated immunity and chronic multifocal oral candidoses.



It can happen on other sites besides the commeasures, it can be on the alveolar ridge…



When u see this lesion ,u think :this is a white nodular lesion that can't be removed by scrapping , so u may think of :

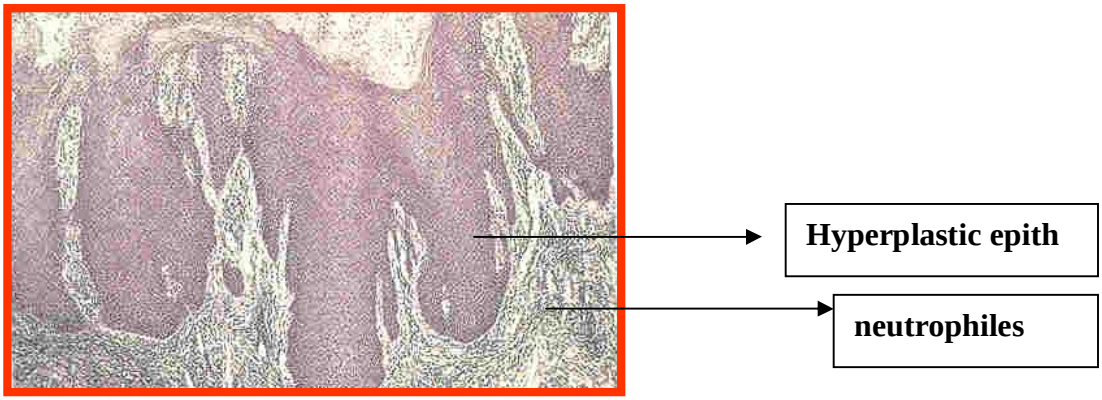
- all kinds of leukoplakia among which candidal leukoplakia.
- SCC :squamous cell carcinoma
- Carcinoma in sito …or any other possible diagnosis

✳Histo of candidoses✳

Look at the pic below ,

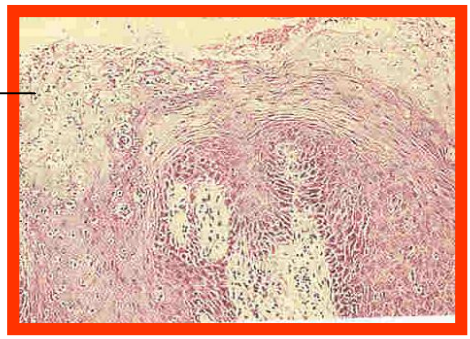
- the epith is considered hyperplastic ;meaning there is increase in thickness of epith,
- and these black dots are neutrophiles in the submucosa going up through the full thickness of epith trying to reach the hyphae,,
- we have hyphae only on the surface …. "in PAS statin"
- we'll have inflammatory infiltrate in the submucosa that will accumulate there and sometimes may form a **small abscess** ,

what's an abscess ? it's a collection of neutrophiles and dead RBCs and desquamated epith cells



if we have a collection of neutrophiles within the epith, we call it a microabscess ,and if I see this microabscess microscopically, I will ask for a PAS stain and I will try to find the hyphae there.

Neutrophils Microabscess



PAS stain

Parakeratin layer

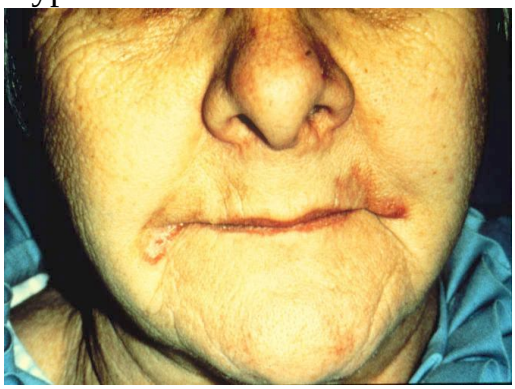
Hyphae in the prickle cell layer

Neutro -philes going up.

◈angualr chilitis:

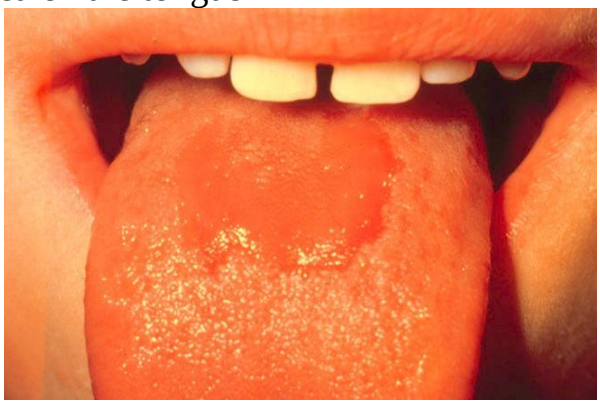
- Is an infection of the cornenrs of the mouth ,
- may be caused by cand or bacteria
- is found in pnts who wear dentures and have low vertical diminution why? Cuz saliva will pool there facilitating colonization of cand or bacteria or both; this pooling may be due to a habit or other causes besides denture wearing . any of which may cause angular chilitis.
- if it doesn't respond to antifungal ,it can be bacterial in origin ,so it needs a facitic acid "antibacterial" or it can be due to combination of bacteria and fungus ,so it needs both antifungal and antibacterial .

In the pic below we have an old man with low vertical dimention ,notice the redness, ulcerations, whitish areas ,fissures ,cracks ,crustings ,painful areas deep folds, the causes are pooling of saliva, may be nutritional deficiencies; some pnts who have B١٢ deficiency, iron deficiency or folic acid deficiency; their epith surface will not be well formed and will be easily affected by hyphae.



◈median rhomboid glossitis:

- Median : in the middle
- Rhomboid: it looks rhomboid “ma٣een”
- Glossitis: red area on the tongue



- It’s not caused by cand, but may be ASSOCIATED with cand
- we have theories about its etiology; it could be a developmental lesion :they say that it’s because that in front of the foramen secum, the filifrm papillae failed to form.
- some and not all of the pnts had hyphae in their smear, so it may be a lesion associated with candidoses .
- u may have an opposing lesion on the palate as we saw before, so we may have a multifocal cadidoses if we have lesions on both palate and tongue.

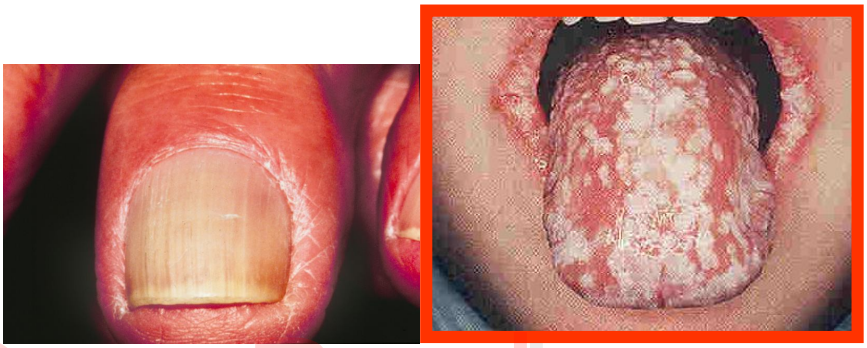
◈CMC “chronic mucocutaneous cadidoses”:

♬ This disease occurs in pnts with impaired cell mediated immunity

♬ these pnt have superficial infection on their skin by cand, having maculaes with scaly whitish surface that can be scrapped.

♬ they have nail bed infections which will make sloughing in layers .

♬ and intraoral candidal infections most commonly **candidal leukoplakia** and it’s multifocal.



❗QUIZ❗

❗ give me the names of candidal infections we had so far?

❗ Acute psuedomembranous candidoses “thrush”
❗ chronic psuedomembranous candidoses “in AIDS”
❗ Acute erythematous “atrophic” candidoses “antibiotic sore tongue”
❗ Chronic erythematous “atrophic” candidoses “candida associated denture stomatitis”
❗ Chronic hyperplastic candidoses “candidal leukoplakia”
❗ Angular chilitis
❼Candida associated lesions “median rhomboid glossitis”
❗ CMC

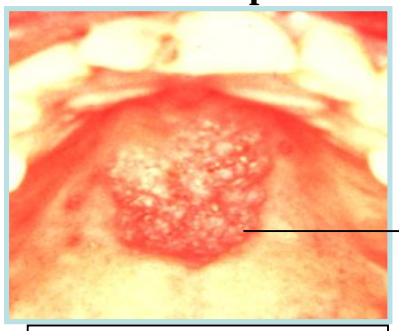
❗ What’s the form of cand. That is found in infected lesions??
Hyphae

❗ What’s the condition affected by candidal infections but doesn’t have hyphae upon biopsy; only minimal or no hyphae at all??
Candida associated denture stomatitis

✳deep fungal infections✳

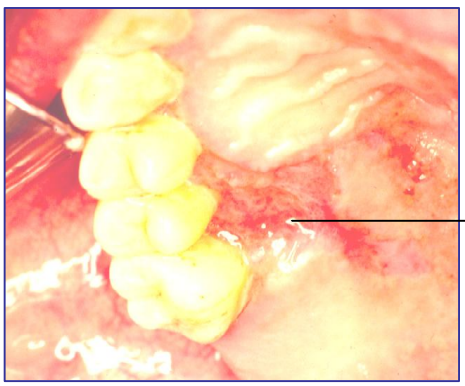
- All the **candidal** infections are SUPERFICIAL infections.
- the deep fungal infections are rare.
- usually occur as systemic infections.
- Deep: meaning that they first for example go to the lung and then reach the oc and induce oral lesions there , so the oc is not the primary site it's usually the secondary .
- does candida cause a deep fungal infection ? **NO**
  Deep fungal infections are cause by another group of fungus.
- examples:

Histoplasmosis **zycomycosis** **blastomycosis**



blastomycoses

Exophitic growth on the palate with whitish and red areas , we may think of SCC or other malignancies before considering deep fungal infections ,so, the biopsy here is important for our diagnosis



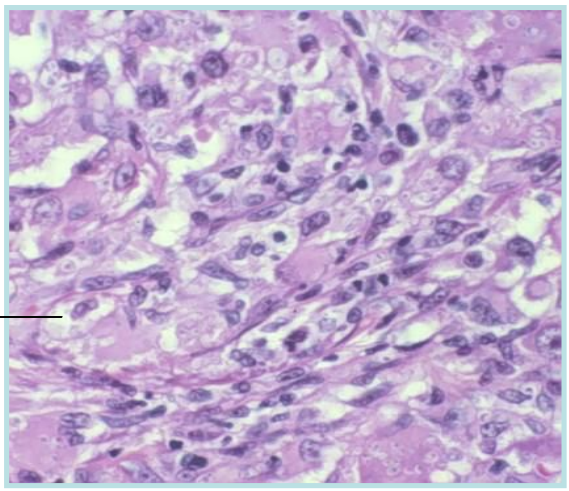
histoplasmoses

Ulcer : why could we have ulcer on the palate without a traumatic cause??

- ☛ we assume recurrent intraoral herpes ; but I don't see pinpointed lesion ,
- ☛we may think of cytomegalovirus infection cuz we said it produces nonspecific oral ulcerations ,
- ☛we may think of SCC and other types of malignancies ,

but once u take a biopsy to establish the dignosis u will be surprised that it's a deep fungal infection

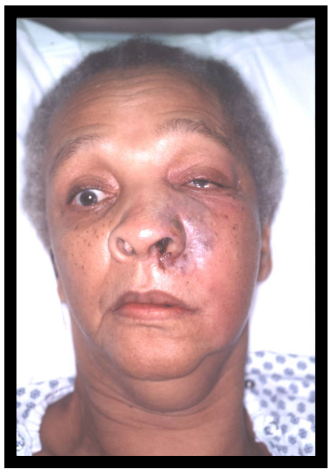
Macrophage with a kidney shaped nucleolus and if u look carefully u'll notice granules in the cytoplasm which are "histoplasmosis fungus"



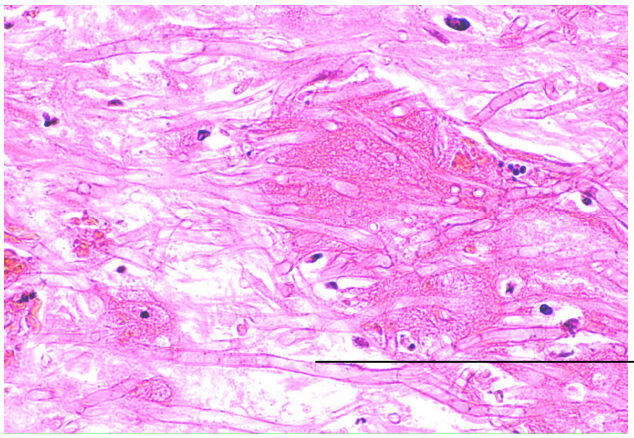
Histo of histoplasmosis : here we see big cells which are macrophages engulfing small particles which are hisoplasmosis fungus , so we ask for GMS stain and PAS stain to establish our diagnosis .

zycomycosis

- it's a deep fungal infection ,here in the picture it affected the maxillary sinus and spread to the eye and the brain.
- it comes from mold "٣fan" ,may be from molded bread or bad vegetables and fruits , here the pnt is susceptible cuz she had diabetes, so she got infected.



This pnt used to be in our clinic and she had diabetes, and zycomycoses prefers the ketoacidoses and the products of metabolism that occur in diabetic pnts ,
even though she had I.V antifungal , she died ,cuz zycomycosis spraed to the brain and it should have been removed by curettage and then we can give I.V and do irrigation



Histo of zycomycoses :Look at these elongated rods ,these are the hyphal form of fungus , these may block an artery and may cut the blood supply and induce necrosis , so we'll have focal areas of necrosis in the brain or the area affected by these organisms .

U need to know the names of these deep fungal infections and the difference in the clinical appearance btw these diseases and those of candidoses .

✳HIV and AIDS✳

What will happen when the pnt gets HIV?

✍Seroconversion within ٣ moths ; the pnt will stay symptomless for ٣ months , in this period the pnt will produce antibodies against HIV in his serum and this's called seroconversion .

✍some pnts will have acute symptoms right away they will have diarrhea , fatigue , fever, weight loss and malaise

✍after ٣ months of seroconversion the pnt will have lymphadenopathy which is generalized and persistent; PGL : persistent generalized lymphadenopathy .

✍after that immediately the pnt will develop the ARC :aids related complex ,with the features that represent the immunodeficiency , such as: Infections bacterial , viral and fungal and Malignancies .

✍What happens is that HIV will bind to:

✈ CD٤ positive T lypmhocytes

✈ and to the macrophages; and this will help spread the infection, it will go from a lymph node to another or within the tissue itself.

✈ and most importantly to CNS cells immediately

✍When the number of CD٤ +ve goes down, the immunity will decrease, and later on we'll have a lot of viral infections , fungal infections and encapsulated bacterial infections causing pneumonia and other diseases.

✍**Most common manifistation itraorally is the oral candidoses** .

✍general differences btw candidoses in AIDS pnts and nonAIDS pnts:
the AIDS pnt :
✈Doesn't respond to antifungal thereby.
✈the clinical picture of candidoses is Multifocal and extensive unlike nonaids who usually has localized candidoses .
✈healing is very difficult , in nonAIDS healing is within ٢ weeks .
✈he may have different forms of candidoses together hyperplastic with pseudomembranous…

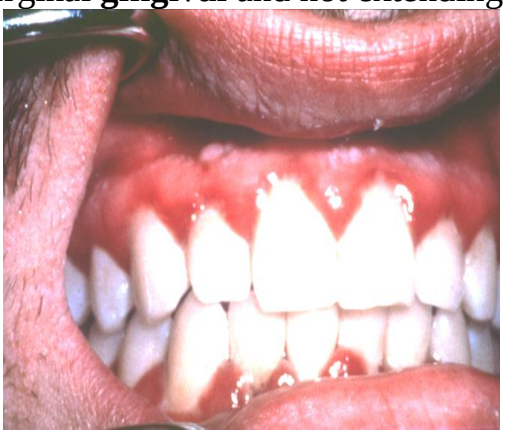
✍The most common candidal infection in AIDS is **chronic pseudomembranous and erythematous** , these are resistant to antifungals and are multifocal and may involve any part of the oral mucosa.

✍٧٠٪ of aids pnts have oral candidoses while only ٢٠٪ of those with seroconversion, they still have some good immunity but with time as CD٤ +ve T cells decrease candidoses increases.

◈HIV related peridental diseases :

١- linear gingival erythema

✍In arabic : "٥a٦ ٢٧mar on gingiva" this line is continuos,
Affecting only the marginal **gingival** and not extending to the attached one.



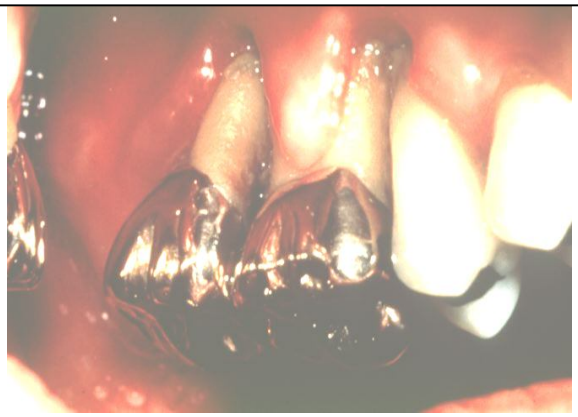
✍ when u see this line, U may think of plaque, calculus and poor oral hygien,e but these pnts have a very good hygiene, so why do they have this disease ??

The theories say: it may be a hypersensitivity reaction to cand that is colonizing the mouth, so it has been associated with **cand.A.**

✍U shouldn't mistake it with ANUG, although like ANUG it only involves the marginal gingival, but there is no necrosis and no loss of interdental papilla and no replacement with grayish membrane.

✍this disease is not responsive to conventional gingival therapy

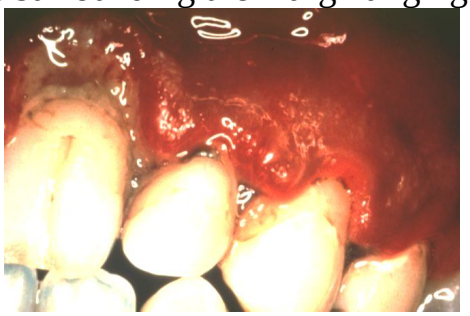
٢-acute necrotizing ulcerative PERIODONTITIS ANUP



✍This is not ANUG , this's concerned in the periodontium and not gingiva only.
✍Its characteristic feature is that it may be **LOCALKIZED.** everything will be okay except for ٢ molars that will have loss of the gingiva, loss of the bone, exposed roots and the pnt already has excellent oral hygiene .
✍there will be aggressive or rapid destruction of the periodontal tissue in the localized areas .
✍the cause is severe impairment of local defensive mechanisms like CD٤ +ve cells against microorganisms in the oc
✍ANUP is not responsive to conventional periodontal therapy .

٣-ANUG

✍ANUG may be found in AIDS pnts, cuz one of the predisposing factors of ANUG is decrease in the function of the immune system, and u can see the greyish membrane here surrounding the marginal gingival.



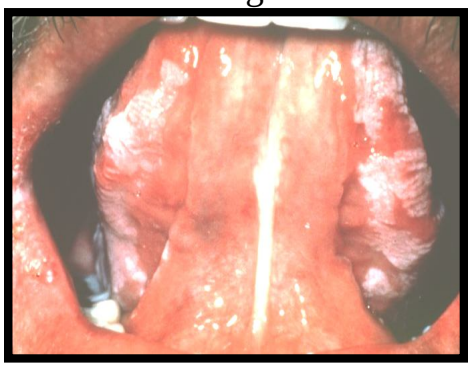
٤-hairy leukoplakia

✍it's caused by EBV.

✍the virus will be in the epith of the pharynx or the B cells.

✍It will get access to the epith at the lateral borders of the tongue ,why ? cuz it's more prone to trauma due to its movement , so there will be a rapid access of EBV there.

✍It may have a smooth surface ,so, it doesn't have vertical folds all the time , it may have vertical folds and hairy appearance or smooth white surface on the lateral borders of the tongue.

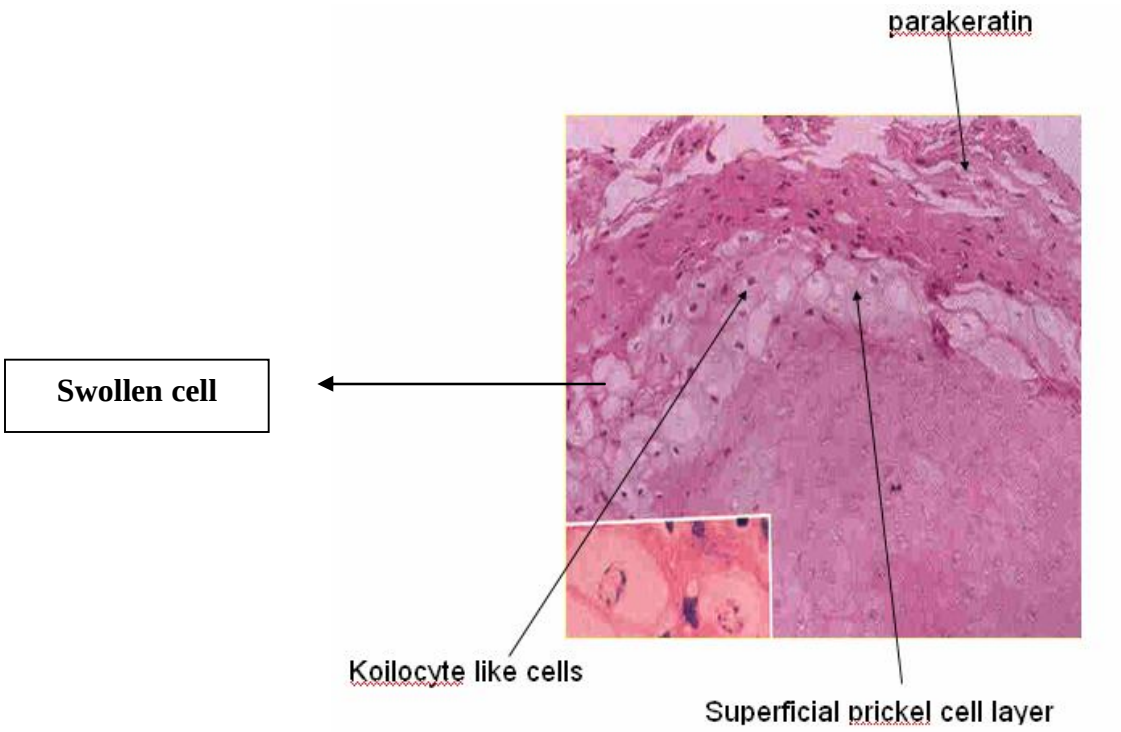


✍it's an Opportunistic infection .

✍**The most important feature is Marked reduction of langerhan cells in the mucosa**; meaning the immune system is not working well allowing the EBV to enter, so any pnt "not only AIDS" with impairment of langerhan cells will have EBV like transplantation, corticosteroids and cytoxic medications … or any cause suppressing the immune system.

✍**It's NOT premalignant ,** it's only a viral infection.

✍Microscopically we'll see :
🗝Acanthosis and increase in thickness of prickle cell layer
🗝parakeratosis and peknotic nuclei
**these ٢ features give the wide appearance "parakertosis and acanthoses" at the superficial layer or the prickle cell layer u'll see
🗝 swollen cells with prominent boundaries and fragmented chromatin due to the replication od EBV
🗝swollen cytoplasm
**These give the appearance of edematous like prickle cell layer

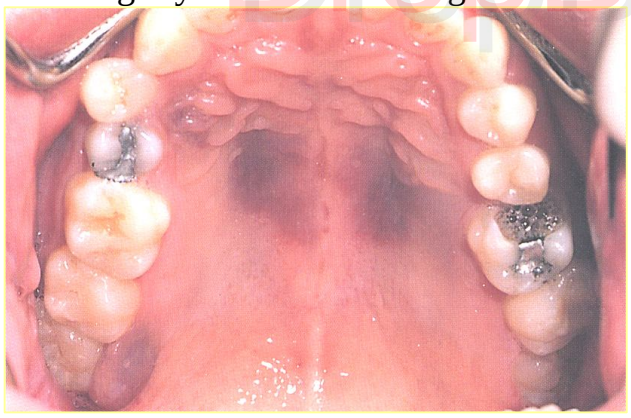


٥- Kaposi sarcoma

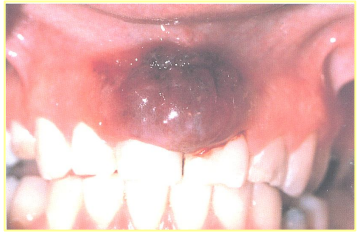
- **It's the commonest tumor affecting the AIDS pnt**
- it's a malignancy affecting the endothelial cells

Candidal infections are the most common infections affecting AIDs pnts
Kaposi sarcoma is the most common malignancy affecting AIDS pnts

- there's an association with the human herpes virus ٨ ;HHV ٨
- could be multifocal.
- intraorally it occurs on the **palate and gingiva**
- and extraorally at **the tip of the nose**.
- Notice in the picture below, here u see a plaque like area, plaque is an area Slightly raised with change in color .



- below u see a nodule which is a raised area, larger than ٥ mm in diameter.

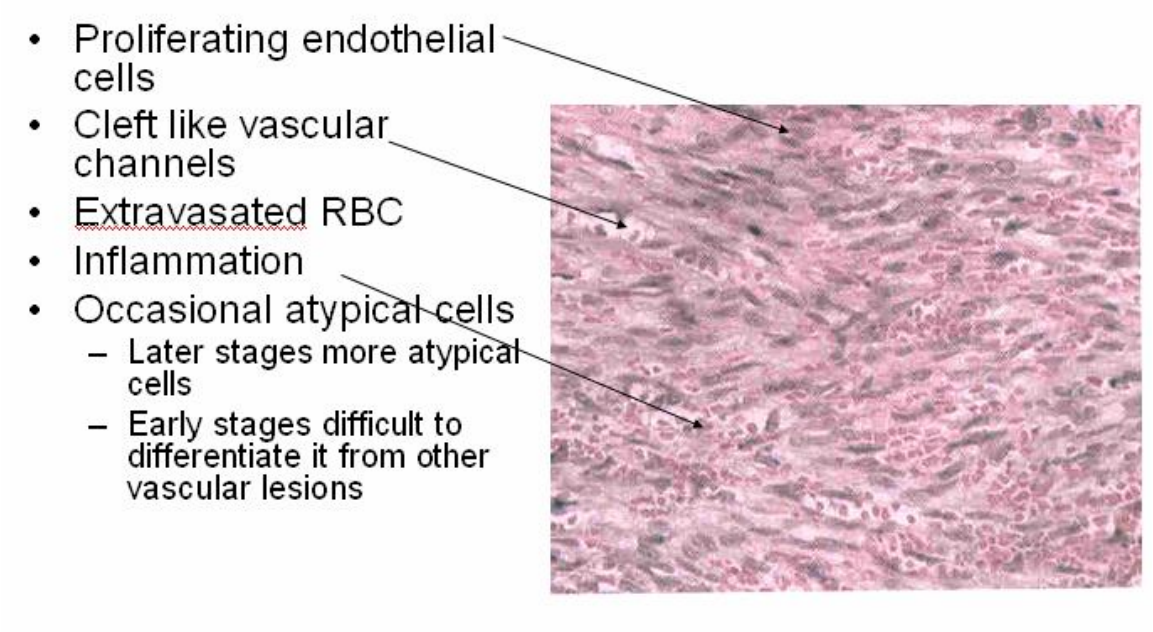


🕊So, in Kaposi sarcoma we may have a plaque, macule* or nodule.
**macule is a smaller area than the plaque .

🕊multiple lesions may be present there

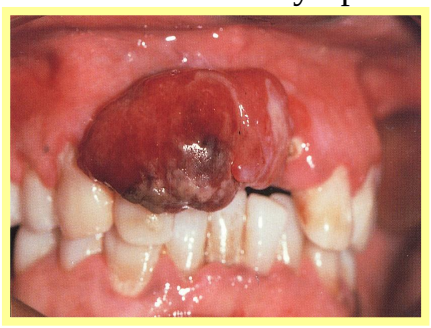
🕊it may be locally destructive, but dosen't need the treatment of say angiosarcoma cuz it's only removed locally.

🕊histo:
🗝here, all these cells are Malignant endothelial cells
🗝The red cells are RBCs
🗝The cells are hyperchromatic "when the cells are black"
🗝the cells are Polymorphic
🗝 slit-like spaces
🗝In Kaposi we don't have well formed RBCs



٦-Lymphomas

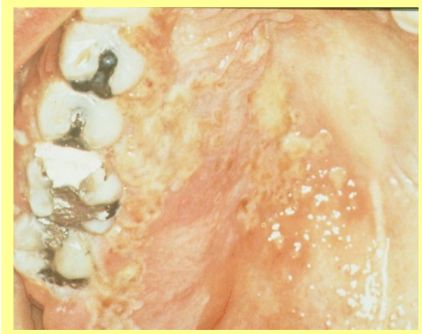
- the most common lymphoma in HIV pnts is the **nonhodgkin's lymphoma**.
- We said nonhodgkin's starts outside the lymph nodes or extranodally



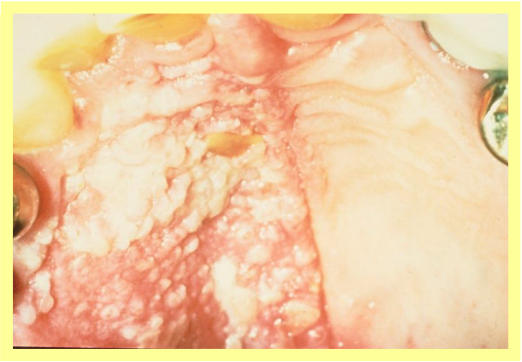
٧-the HIV goes to the CNS causing neurological disturbances and facial nerve palsy .

٨-viral infections

- we have HSV , cytomegalovirus, HZV…
- these will produce atypical ulcerations, in normal pnts ;the HSV ulcer was pinpointed but here it's widely spread with sloughing.



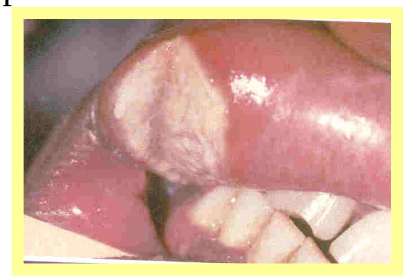
HSV



HZV :causing ulcer in the palate

٩-ulcers

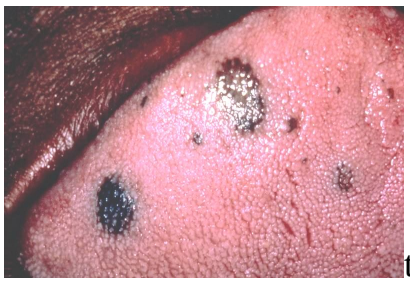
Sometimes AIDS pnts have ulcer that are not associated with any virus or any cause it's just a nonspecific ulceration.



١٠-Salivary gland disease

HIV pnts have **lymphepithelial cysts ,** they will have enlarged salivary glands due to lymphocytic infiltrate, mostly the parotid gland.

١١-idiopathic thrombocytopenic purpura

 this is an autoimmune response …

The end.
Done by : ESRA'A DARAGHMEH

…… !!